and or said T-cell is] providing said individual with a cell comprises providing a cell derived from said individual.

27. (Amended) [Use of an dendritic cell according to claim 16 in a treatment for] A method of treating an individual suffering from a disease selected from a group consisting of an auto-immune disease, an allergy, a graft versus host disease, [and/or] and a host versus graft disease, comprising:

providing an isolated dendritic cell capable of functionally modifying a T-cell specific to an antigen such that the response of said T-cell to said antigen is altered; and

introducing said isolated dendritic cell into said individual.

Please add the following new claims:

28. The method according to claim 1, further comprising incubating said dendritic cells with cell homogenate containing at least one antigen of interest before activating said dendritic cells in the presence of a glucocorticoid hormone.

29. The method according to claim 13, wherein providing said dendritic cell with a glucocorticoid hormone comprises providing a precursor of said dendritic cell with said glucocorticoid hormone in vitro.

Please cancel claims 2, 3, 21 and 23 without prejudice or disclaimer.

Remarks

The application is to be amended as previously set forth. The changes are generally made to correct minor typographical and format errors and to more appropriately claim the invention in view of United States practice. As indicated, substitute pages 1a, 1b, and 4a-4c are enclosed herewith in order to simplify amendment of the application. It is respectfully submitted that no new matter has been added.

Conclusion

In the event questions remain after consideration of these amendments, the Office is kindly requested to contact applicant's attorney at the number given below.

Respectfully submitted,

Allen C. Turner
Registration No. 33,041
Attorney for Applicant
TRASK BRITT, PC
P. O. Box 2550
Salt Lake City, Utah 84110-2550
Telephone: (801) 532-1922

Date: September 21, 2000

Enclosures: Substitute pages 4a-4c

BRIEF DESCRIPTION OF THE DRAWINGS

Fig. 1 Pretreatment with DEX inhibits the phenotypic changes induced by CD40 ligation.
Seven days immature DC were cultured for 24h in the absence or the presence of 10^{-6}M DEX and activated via CD40 with the CD8-CD40L fusion protein for 48h. The comparison with immature DC maintained in medium alone is shown. Empty histograms show the background staining with isotype controls MoAb and solid histograms represent specific staining of the indicated cell surface markers. Specific mean fluorescence intensities are indicated. Mean fluorescence intensities of isotype controls were between 3 and 4. Data are representative of 4 independent experiments.

Ab cont

Fig. 2 DC triggered through CD40 maintain an activated phenotype upon a subsequent DEX exposure.
Immature DC were activated with the CD8-CD40L fusion protein. DEX (10^{-6}M) or medium control were added 48h later and cells were analyzed after 2 additional days of culture. The comparison with immature DC maintained in medium alone is shown. Empty histograms show the background staining with isotype controls MoAb and solid histograms represent specific staining of the indicted cell surface markers. Specific mean fluorescence intensities are indicated. Mean fluorescence intensities of isotype controls were between 3 and 5. Data are representative of 2 independent experiments.

Fig. 3 Pretreatment with DEX does not affect the regulation of DC antigen uptake machinery.
Immature DC were incubated in the absence or the presence of

10^{-6}M DEX for 24h and further activated or not via CD40 with the CD8-CD40L fusion protein for 48h. Cells were pulsed for 1h with medium containing either 1mg/ml FITC-BSA or 1mg/ml FITC-mannosylated BSA. Empty histograms show the background autofluorescence, Grey-filled histograms show the background uptake at 0°C. and black-filled histograms show the specific uptake at 37°C. Data are representative of 3 independent experiments.

Fig. 4 Pretreatment with DEX alters the cytokine secretion profile of CD40-triggered DC.
DEX-exposed or control immature DC were left in culture without further treatment or stimulated with the CD8-CD40L fusion protein. Culture supernatants were harvested 48h later and IL-10, IL-12p40 and IL-12p70 secretion were analyzed by specific ELISA. Data are representative from 6 independent experiments.

Fig. 5 Pretreatment with DEX impairs the T cell stimulatory capacities of DC activated via CD40 and leads to a state of hyporesponsiveness of Th1 cells.
Allogeneic MLR: non adherent allogeneic PBMC were cultured with different numbers of CD40-triggered DC, DEX-treated CD40-triggered DC or immature DC. The proliferative response was measured on day 5.
Th1 stimulation assays: Hsp65-specific T cells were cultured with different numbers of HLA-DR matched CD40-triggered DC or with DEX-treated CD40-triggered DC pulsed with the hsp65 protein or with the specific p3-13 peptide epitope. The proliferative response and the T cell dependent IFN-g production were analyzed on day 3. Data are representative of 4 independent experiments.

Fig. 6 DEX-treated DC triggered through CD40 induce a state of hyporesponsiveness in Th1 cells. Hsp65-specific T cells precultured with CD40-triggered DC or with DEX-treated CD40-triggered DC pulsed with the p3-13 peptide epitope were harvested after 48h, allowed to rest in the presence of 5U/ml IL2 for 3 days, and restimulated with p3-13-pulsed DC. The proliferative response and IFN-g production were measured on day 3. Similar results were obtained in 2 independent experiments.